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EDITORIAL

PAIN

by FREDERIC R. STEARNS, M.D., *Editor*

While pain is a most important protective mechanism of the human organism, it is also one of the most dreaded symptoms in all pathological reactions. Thus, the physician is concerned with pain from two aspects; as a signalizing sign, frequently pointing to the site and character of the pathology, and as a symptom which, in most cases requires, immediate attention and care.

This first editorial on pain shall deal essentially with the physiology of pain. It is difficult to give a satisfactory definition of pain. The sensation of pain is a rather explicit one; and a majority of individuals describe the perception of pain in various abnormal reactions in a like manner (burns, abdominal pain in appendicitis, pain in angina pectoris, pain in pleurisy, in trigeminal neuralgia, etc.); in other abnormal states the descriptions vary such as in headache, stomach ache, and in most conditions which give rise to "referred pain." Furthermore the threshold of pain sensation varies not only from individual to individual, but also in the identical individual from situation to situation. Santt and Fleischmann have stressed that the pain threshold diminishes after the first experience owing to conditioning; the less intense stimuli which were not previously reacted to as painful become signals (conditional stimuli) of the stimulus which is to follow and they are reacted to as if they were painful. We also know that histamine which plays a powerful role in the biochemistry of the organism lowers the threshold of pain (Thomas and Butler). On the other hand, it is a long observed fact that emotional disturbances may increase the pain threshold. It is possible to be inattentive and to disregard pain in the violence of a fight or in emotional tension. The same imperception may occur in mentally defective persons, in religious exaltation, in hysterical states, in the trance of mediums, etc. (Critchley). Strauss, thus, was justified in stressing that pain is an emotional experience, depending on the individual's interpretation of bodily sensations. Neither its psychic nor its somatic components may be considered solely responsible. The importance of the emotional aspects with respect to the somatic side is evidenced by the reaction to pain in patients after prefrontal lobotomy; they experience pain in the same manner as "an undomesticated animal." Physiologically one may differentiate between pain of the peripheral and pain of the sympathetic type. As to the first kind, the knowledge concerning the manner in which pain impulses are formed is incomplete. Pain sensation apparently originates in the stimulation of free nerve terminations in the skin. After the fibers, carrying pain sensation, enter the posterior gray column in the spinal cord, a secondary relay of axons crosses to the opposite site in order to ascend as the lateral spinothalamic tract. Aft-

EDITORIAL

er passing the mesial fillet the axons reach the thalamus and proceed from there to the parietal cortex ending in the parietal convolution which lies posterior to the central Rolandic sulcus. (Wilson and Mussey). Production of peripheral pain seems to be associated with local chemical changes within the abnormal tissue foci. Apparently the tissue pH may alter toward acidity or alkalinity. Factors such as time of the day and food intake affect the acid-base balance and may influence the intensity of pain. In comparing urine pH with the degree of pain, felt by patients, two distinct (acid and alkaline) patterns were found. Administration of alkalinizing agents increased alkaline pain and relieved acid pain, and vice versa. The action appears to bear directly on the abnormal foci rather than on structures conducting the pain impulse since both patterns may coexist in the same individual. (Revici and others). Regards sympathetic pain, Leriche has differentiated it aptly from the peripheral, or as he terms it, cerebrospinal type. Sympathetic pain is more diffuse, of a spreading type, irregular in distribution, and essentially affected by, or affecting, the emotional state, while peripheral pain is localized with precise anatomical limits (Hamblen and Cuyler). The stimulus of sympathetic pain is probably conducted to that portion of the spinal cord from which the nerves that supply the organ in question originate. From there the stimulus may be distributed as unpleasant sensation or real pain to the somatic area served by the segment of the cord concerned or may be felt as a generalized discomfort. (Wilson and Mussey). The problem of sympathetic pain leads to that of referred pain. Sinclair, Weddell and Feindel have explained the mechanism of referred pain in postulating that an important factor in its production is the existing of branching among the sensory pathways conduction sensation of pain. This branching of axons from the site of the stimulus to other not involved sites leads to misinterpretation by the central nervous system of the true origin of the pain and, by liberation of metabolites at the nerve terminals in the region of the pain stimulus, secondary pain impulses are produced. An impressive example, of referred pain is, the so-called viscero-cutaneous pain arising from painful stimuli in the diaphragm, heart, esophagus and stomach which are mediated over visceral afferent neurons. (C. L. Dana).

All these peripheral and sympathetic pain stimuli are integrated in the central nervous system. Three levels of intergration have been recognized; the lowest is the tectum of the mesencephalon; the second is situated in the posterior ventral nucleus of the thalamus, and the highest is located in the cerebral cortex. In the cortical endings of the pain pathways, the integration of the sensation of pain to the feeling of pain takes place. The perception at the cortical level is, aside of the original pain stimulus, also determined by other qualities of superficial and deep sensibility and, furthermore, amplified or inhibited by the activity of other cortical centers. (Krayenbuhl and Stoll).

In a following editorial the pathophysiology of pain shall be touched upon.

ORIGINAL ARTICLES

Drug Hazards in Cardiovascular Diseases

by NATHAN FLAXMAN, M.D.

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The introduction of new and diffent forms of drugs has led to more difficulties in the treatment of cardiac disturbances. A great deal of trouble seems to arise because many potent drugs are being used routinely. The theme song seems to be: it's new, it's harmless, so why not use it. However, not all reactions are based on this fallacious idea. Many occur with the use of the time-tested cardiac drugs. For these reasons a review of the more recent problems arising from the use of such drugs is presented. Most of the dangers arise from (1) digitoxin, (2) mercurial diuretics, (3) quinidine, and (4) dicumarol.

Digitalis and Digitoxin

In the past ten years digitalis poisoning has become common.[1] The increased frequency seems to coincide with the shift to the glucoside preparations, especially to digitoxin, and the routine methods advised for the use of this drug. Further difficulty has arisen because the common symptoms of anorexia, nausea, and vomiting, known to be due to digitalis overdosage, occurred infrequently in patients treated with digitoxin. Instead, signs of disorders of the cardiac mechanism, expecially the more serious conduction disturbances as auricular fibrillation, A-V block, sinus arrest, and aparoxysmal ventricular tachycardia, were the earliest and most frequent clinical and electrocardiographic manifestations of digitoxin posoning.

When properly given digitalis yields good results. Its action is primarily on the myocardium, increasing the efficiency of the heart as a pump. Through this action, the refractory phase is prolonged and conduction is slowed through the A-V bundle. As the dose of digitalis preparation, and this applies especially to digitoxin, is increased, the direct action on the A-V conduction becomes important. As conduction is slowed, digitoxin increases the irritability of the myocardium. This results in abnormal impulses from ectopic foci, and, if the irritability is increased an abnormal mechanism may replace normal conduction. Such action of digitalis can frequently be avoided by individual therapy with the whole-leaf preparations. Digitalis is indicated in congestive heart failure and auricular fibrillation. When the indication for digitalis exists, there are no contra-indications.

During the past year more hazards appeared along these lines. Caution has been urged with the use of digitoxin, even with a dose of 0.05 mg. daily.[2] This drug caused ventricular tachycardia with peripheral vascular collapse following the use of a single oral dose of 0.6 mg., an extremely serious complication. Four cases of digitoxin intoxication,[3] one with ventricular tachycardia, were reported.[4] Increasing congestive heart failure developed as a manifestation of digitalis toxicity.[5] Nodal tachycardia

followed digitalis overdosage in two instances and was stopped by carotid sinus pressure.[6] Chronic effects of digitalis intoxication, manifested by diarrhea and auricular flutter, also were reported.

Mercurial Diuretics

The use of mercurial injections in the treatment of congestive heart failure has inestimable value. However, routine daily administration has produced some serious reactions. The untoward effects have been emphasized.[8] Acute urinary retention,[9] the salt depletion syndrome,[10,11] and acute dehydration[12] occurred. The intramuscular mercurial diuretic, Mercuhydrin, caused a fatality[13] and a sensitivity reaction.[14] Undesirable effects are (1) reactions due to diuresis with or without changes in the electrolyte balance; (2) reactions related to the mercury portion of the molecule; and (3) reactions related to the organic structure of the specific mercurial compound. It is important that the salt intake, sodium chloride and not a salt substitute, be maintained to prevent the sodium depletion syndrome. By digitalizing the patient first, by allowing fluids and salt as desired without any restrictions, by giving the amount of mercurial as needed, and by keeping in mind that spontaneous redigitalization can occur, the hazards in the use of mecurial diuretics can be avoided.[15]

Quinidine

The drug of choice for the therapy of ectopic rhythms of the heart is quinidine.[16] It is strongly advised in (1) fresh auricular fibrillation or flutter with a normal roentgen shadow, normal heart sounds and normal blood pressure, (2) ventricular tachycardia, and (3) post-thyroidectomy flutter or fibrillation. It is generally accepted in (1) relatively old flutter and fibrillation, (2) ventricular premature beats, (3) paroxysmal or nodal tachycardia which fail to respond to other measures, and (4) auricular fibrillation occurring during myocardial infarction. Although evidence in its favor is small, it is used as a prophylaxis in myocardial infarction to prevent arrythmias.

Quinidine is strongly contraindicated in (1) complete heart block, (2) bundle branch block or intraventricular conduction defect, (3) subacute bacterial endocarditis, and (4) over-digitalization. It is generally rejected in (1) congestive heart failure, (2) auricular fibrillation which has replaced angina pectoris, (3) hyperthyroidism with sinus tachycardia, (4) markedly enlarged heart, and (5) severe mitral stenosis. Although the evidence of danger is small, it is often rejected because of (1) risk of embolism from the auricles, or (2) rise in the ventricular rate in auricular flutter.[16]

The hazards emphasized in the literature in the past year were quinidine allergy[17] and thrombocytopenic purpura.[18,19]

Dicumarol

The routine use of dicumarol in the treatment of congestive heart failure, as well as in acute myocardial infarction, has been advised.[20] A number of reports showing its hazards has accumulated. Hemopericardium without rupture of the heart caused death following dicumarol therapy for myocardial infarction.[21] Five deaths were described as complications of this an-

ticoagulant therapy.[22] A fatal retroperitoneal hemorrhage followed lumbar sympathetic block during treatment with dicumarol.[23] The drug caused a fatality in severe hypertensive and arteriosclerotic cardiovascular disease despite controlled therapeutic levels.[24] Death occurred due to dicumarol in long-term therapy to prevent recurrent coronary artery thrombosis.[25] Two hemorrhagic episodes of major severity,[26] major bleeding from a single dose of dicumarol,[27] and cardiac tamponade[28] also occurred.

Khellin

In the treatment of angina pectoris with preparations of Khellin (amni visnaga), there were uncomfortable side effects, namely, nausea, anorexia, and dizziness in about 60 per cent of patients.[29] These were real deterrents to the use of the drug in many instances.

Summary

Certain hazards exist in the use of drugs for the treatment of cardiovascular diseases. A review of the literature for the past year shows that these have arisen mainly from the use of (1) digitoxin, (2) mercurial diuretics, (3) quinidine, and (4) dicumarol. All are useful drugs in the treatment of cardiac disturbances. Digitalis intoxication has become common, most frequently with the use of digitoxin. The administration of any digitalis preparation required individual prescribing. Symptoms of digitoxin poisoning are infrequent, as it is manifested far more often by signs of conduction disturbances.

Routine injections of mercurial diuretics in the treatment of congestive heart failure is not advised. It is a dangerous procedure in elderly cardiacs. Acute urinary retention, acute dehydration, salt depletion, and uremia may result. Salt should be given freely to cardiacs being treated with mercurial diuretics.

The indications and contraindications are given for quinidine, still the drug of choice in most ectopic rhythms. Quinidine allergy and thrombocytopenic purpuras have been reported.

Dicumarol administration should not be a routine in the treatment of congestive heart failure or acute myocardial infarction. Whether or not the laboratory control of prothrombin time determination is perfect, or a single dose only was given, or long-term therapy used, the danger of hemorrhage is always present. Nine dicumarol fatalities have been reported in the past year.

In the treatment of angina pectoris khellin causes uncomfortable side effects, such as anorexia, nausea, and dizziness in a majority of such patients.

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ORIGINAL ARTICLES

The Diagnostic Dilemma

by RICHARD D. LOEWENBERG, M.D.
Bakersfield, California

Every experienced physician must have wondered why he feels so involved in his diagnostic troubles. As in all other life experiences, the disappointments are the most instructive school in our never ending training. Concern for the patient and for our reputation do not exhaust the issue nor the poor judgment so often mentioned.

The following informal and unsystematic observations are concerned with these everyday occurrences in medical practice. They have found little attention in spite of the vast literature on differential diagnosis: Those fortunate among our profession who never missed a diagnosis nor were ever disturbed about their inevitable pitfalls might skip these notes. This is written for those among us, who have often wondered about the lessons of our errors; hesitating to touch such a delicate subject just as we would hide our blood pressure, our age, or our dreams. This betrays a definite awareness that the doctor as a person is more deeply involved in his medical work than our training in natural science would lead us to expect. He recognizes the significance of this fact well enough when he avoids treating his own family, but rarely goes beyond this, asking whether his own emotional factors might not likewise operate at times with his patients. Can he not trust the assembly line of complete workup with all laboratory tests? Do not group clinics seemingly reduce all loopholes, so that he might comfort himself in the illusion of strict objective methods? What, then, is involved? Can a conscientious, critical practitioner do anything more? Certainly there is no disregard for the modern medical diagnostic methods, which nobody could dare to ignore, however more may be required. The authors on psychosomatic medicine usually present us with a generous list of desirable personal qualifications for the physician without concentrating on the nature of his diagnostic errors. The individual's conscious and unconscious factors are beyond the scope of this paper, in which we have to confine ourselves to demonstrating those general reactions common to all medical work. The psychiatric veiwpoint is here applied as a tool to the physician's distress in order to look for some helpful insights.

Characteristically enough, the cooperation of other doctors was not successful as they knew only of cases of their colleagues, in which I was not interested; therefore, I have to report from my own extensive experience in hospitals and private practice which, over the decades led me from general practice work to the neuropsychiatric speciality. I don't for a moment think that in principle the situation in surgery, obstetrics, pathology, or any other line is much different when it comes to the subjective factors of our own, that are common

to all physicians. I also don't doubt that many other elements, not touched upon here, are just as important. I do not think the ratio of my errors, having been collected from several thousand cases, is larger than that of any other doctor under similar circumstances. I believe, however, that I was not afraid to try to analyze some of the personal factors.

A short condensation of my own case histories (that might easily be increased by any experienced clinician) is given, concentrating entirely on the diagnostic difficulty of being sidetracked into errors.

Case No. 1: In Shanghai, in the middle of August, 1937, two members of the U.S. Fourth Marines got sick with high fever, extreme headaches, and drowsiness lasting one day only. Quick unevetful recovery. These cases are mentioned only because they caused me to miss a diagnosis of a third patient, lying between these patients in the same ward. Within 36 hours a sailor aged thirty-six, was also seized by high fever, cervical rigidity hemiplegia of the left side, coma. The sudden onset, the apparent connection with the two above mentioned cases led me to diagnose a fulminating hemiplegic form of acute Encephalitis, often seen especially in the St. Louis outbreak. The post-mortem, performed 36 hours after the onset of the symptoms revealed a tumor of the right cerebral hemisphere, Gangliocytoma.

This was one of the not so uncommon acute tumor cases often described in literature, in which an asymptomatic incubation period suddenly develops into a fulminating fatal picture of few days' duration.

Case No. 2: (C. H., No. 140764, Kern General Hospital). The patient is a twenty-seven year old, white female. HISTORY: The patient had one convulsion as an infant. At the age of seven, she fell from a swing and broke her nose and was unconscious. In 1944 she had a goiter operation in Los Angeles. The patient has a horror of being with her husband, although she likes him. The day before admission she learned that he was coming home. Her mother hinted that the daughter worked in a drug store and had access to medicaments. Besides, the patient has been on sulfa-drugs for a coid. On the morning of October 20, 1945, the patient got up feeling well, finished breakfast, when she complained about a severe headache. She walked to her bed to lie down, vomited, went into a coma, stertorous breathing and convulsions. PHYSICAL EXAMINATION: Pupils dilated, left larger than right, no light reaction, fundus normal. Respiration, stertorous and loud over the chest. Blood pressure, 160/70. Abdomen normal, no neck rigidity, spastic hyperextension of both arms and legs, wrists rotated to the outside, reflexes hyperactive. All pyramidal signs plus. Temperature 103.8. Laboratory exams: Urine analysis negative. Blood NPN 28, blood sugar 162, red blood count 4,450,000, W.B.C. 16,550, Hb. 86% 13.5 gms. Color Index 0.9. Spinal Pressure 400 mm; spinal fluid 16 WBC per cu mm, with sulf. level 2.6 mg%. Wasserman was anti-complimentary. Gold Curve negative.

COURSE: Under increasing respiratory failure, patient expired four hours after admission.

Although there was a confusing history of self-medicated sulfa drugs during the previous week and an injection for a cold received on the previous day, the sudden onset seemed suggestive for subarachnoidal hemorrhage; the high temperature of 103.8 for enephalitis. The post-mortem showed deep in the left temporal lobe, a massive cavity filled with blood. The classification of the glioma which evidently caused the hemorrhage was, according to the pathologist's microscopical examination: "Astrocytoma with extensive hemorrhage into the left temporal lobe of the brain."—(A. E. Rhoden).

The literature on acute deaths, and especially on coma, is so extensive and well known, that it is hardly necessary to mention it here. The concept of poison as a rather vague undefinable menace, dominates not only the public but also our thinking whenever we are faced with a sudden catastrophe for which we were not in any way prepared. The very many new toxic substances used in industry and agriculture have doubtless been responsible for many obscure fatal "heart" cases. In daily practice we should be constantly aware of the paradox that actual poisonings occur where we never suspect them, and that other causes are most of the time responsible when we are told about poisoning. All of us have seen apparently acute alcoholic intoxications behind which (in spite of the odor) were cerebral accidents, cardiac failures, uremic coma, etc. It is worth mentioning how long a time it took to discover the effect of chronic bromism in a medicament more than half a century in universal use.

The role of a good history, which can never be too detailed, can hardly be overstressed, and yet lead to error. Especially in insurance problems and psychoneurotic reactions, the question of connection is one of the most difficult ones, as the positive and negative case illustrations might show:

Case No. 3: (F. S.) In the late twenties, a chronic tabetic came to the hospital ward. He did not give the permission for the spinal tap which had therefore to be omitted in the diagnostic workup. Five weeks later, he developed on the ward, under our eyes so to say, a typical Charcot joint of the second lumbar vertebra. If he had given his permission for a spinal tap, how could anybody have denied the possibility of a traumatic connection with claims for compensation.

Case No. 4: (F.A. No. 153028, Kern General Hospital). June 6, 1946: An eighteen raye old semicomatose Mexican was admitted in an epileptic seizure. Fainting spells and convulsions dated back to an injury two years previously when he was thrown from a horse and fractured the right leg. Seizures occurred about once a month, preceded by an aura. Physical examination, including neurological findings, was essentially negative. *Admitting Diagnosis:* Post traumatic epilepsy, chronic subdural hematoma, "obviously" due to his fall from a horse. Neurosurgical consultation was requested.

June 14, 1946: Spinal Fluid: Protein 55.4 mg %, otherwise entirely negative.

June 15, 1946: Skull X-ray: (condensed) "No fracture or dislocation. Sella normal. Multiple minute calcifications scattered throughout the brain, make a diagnosis of tuberous sclerosis very well probable. One must also consider multiple calcified areas of chronic granuloma or even parasites."

July 17, 1946: Permission for air studies not forthcoming.

July 22, 1946: A medical consultant from Los Angeles suspected convulsive state, but also tuberculosis or coccidiodal meningitis.

August 5, 1946: Patient temporarily came out of coma. Thrombophlebitis of left leg developed.

August 14, 1946: Patient died.

Post-mortem showed: (Abstract) "Many small gray pearly nodules throughout the brain, also protruding into the ventricles. Sections of these lesions showed a cyst-like structure with a fairly thick capsule containing caseous material, typical of tuberous sclerosis."—(A. E. Rhoden).

When we pause a moment, we wonder how the causal connection with the trauma could have been disproved without the post? The role of dissimulation, the hiding of severe symptoms is perhaps as dangerous a pitfall as is the probably over-rated simulation. During my years as physician in China the stoic attitude of the old generation was most impressive and dangerous for the diagnosis because the calmness never betrayed pain and distress, while the younger generation, educated in the blessings of our civilization was as complaintive as any Westerner. All of us might have seen the effect of placebos, such as an injection of water, and then three days later the patient developed a deep jaundice, arousing us from a comfortable optimism. Refusal of any history causes another problem.

Case No. 5: As Railroad Physisian during the war in a small community in the High Sierras, one night a highly unpopular brakeman was brought in with a back injury. He refused to give any history whatsoever, during the entire stay in the hospital, and there had been no witnesses. No neurological signs. The X-ray made with an old apparatus did not show any pathology, therefore, a functional condition was suspected. A month later, when he was rechecked in a metropolitan hospital with modern equipment, a fracture of the lower lumbar vertebra was discovered. Why information about the details of the accident was refused, has never been cleared up.

Case No. 6: Several years ago, I saw a sixty-five year old lady in San Francisco, who was found unconscious on the floor, after having complained about headaches, nausea, and restlessness the three previous days. She developed hyperpyrexia (to 105), right hemiparesis. Laboratory tests were noncontributory. After three days her condition cleared up and one of the leading neurosurgeons of the Bay Area wrote in his report: "I am in full agreement with you. She *undoubtedly* had a cerebral thrombosis."

A month later, the patient re-entered the hospital in coma and the negative laboratory findings and absence of focal neurological symptoms gave the suggestions of a cerebral vascular lesion in the frontal-parietal region. When she was ready to be dismissed to a nursing home two weeks later, her temperature became hyperpyretic and she expired with terminal pneumonic symptoms. The post-mortem revealed a diffuse chronic encephalitis with perivasculary infiltration. This case illustrates the danger of being guided by the age of a patient too exclusively.

My conclusions are naturally not based on the above experiences alone, but also from decades of hospital work, on the observations of the errors of the many young colleagues with whom I had the privilege of living closely together. Then and there the question will come up, whether there are safeguards for our shortcomings, whether there is a way to face and to overcome some of them in time. Only a diagnosis of our missed diagnoses might guide us towards therapy and prevention of our distress. Impersonal "atmospheric" conditions must be differentiated from personal factors. First of all, it must not be forgotten that we are working in a highly charged, peculiar climate. Our patients often feel responsible for organic conditions entirely beyond their control, but not responsible for their life attitudes which they resist changing. Just the same, it is important that the physician learn to differentiate between those causes which are beyond his personal powers and those within his field of responsibility. The vogue of diagnostic and therapeutic fads cannot fail to influence him. This is particularly noticeable in our language habits: Words use medical men as anybody else. A great number of other pitfalls have been left out, as for instance the influence of epidemic seasons (polio!) and age, especially the involution period which at once influences our thinking and does not explain anything. Nearly every disorder and disease can happen at any life period. Adolescent mental disturbances are sometimes hastily stigmatized by young psychiatrists as schizophrenia; old age disorders as arteriosclerosis and senile dementia. The so-called monosymptomatic neuroses are only existent when a few minutes of examination are devoted to the patient who, only then might impress as a stutterer, an alcoholic, or an impotence. The permanent outside pressure under which the doctor works induces him at times against his intention to hasty decisions and pronouncements to a public that is oversold by unwise press, radio, and magazine propaganda and raises the physician to a priest and magician or a devil who manufactures diseases. None of these causes however, or the many not mentioned involved factors are strong enough to reassure the conscience and dilemma of the physician. Such self understood measures as continous study, encouragement of consultations, untiring rechecks of details in histories, and re-evaluation of his patients' problems need no elaboration here.

It is believed he may be helped, in addition, by the cultivation of two apparently opposite forces in him: Resistance and Enthusiasm, to maintain his emotional balance on which

are incessantly made superhuman demands. It is not a matter of the popular "peace of mind" but balanced forces. Whether a doctor knows about it or not, he cannot exist without some kind of personal outlook on life. When a doctor told Chesterton's Father Brown: "I am a practical man, and I don't bother about religion and philosophy", he answered: "You never will be one until you do."

The doctor has to resist, at times, the temptations of his own intuitive snap diagnosis. Even though the first impression may be of great importance and significance, he cannot rely upon it, and might need half a year's patient study for scientific verification. He has to resist both extremes in his daily life: The over-concern of anxiety and anticipation; and the over-confidence of wishful thinking. The dangers of dissimulation are perhaps greater than those of simulation and the resistances against the pressure of a frightened panicky public require constant watching. The temptation of singular dominant symptoms of impressive laboratory findings, the neglect of the picture as a whole, his own preoccupation with another dramatic case easily surrender him to hasty decisions. "Never cease to doubt the correctness of your diagnosis", was a never forgotten warning of one of my clinical teachers. But this attitude of scepticism must be balanced by the original impulse which induced most of us to devote our lives to medicine: the desire to help the suffering and the overpowering urge to learn about the nature of health and disease. The physician's position in these days is wider and more exposed than ever and so are his real responsibilities he must be prepared to shoulder. It is regrettable that the present political fights of our profession do not recognize that the real dangers are the shifting of personal responsibilities to the huge anonymous mass organizations of bureaucracy. Every new patient is a new problem which can never be done justice by comfortable standard practices and rigid formulations or uniform management, which may have proven their value in acute physiological emergencies, but are entirely inadequate in subacute and chronic problems, especially of a psychosomatic nature. It takes all our endurance and enthusiasm to recognize the uniqueness of each new challenge.

In conclusion, we may sum up the lessons we are learning from our diagnostic dilemma: The physician cannot avoid reflecting on his own psychology which his patients are sure to study. The distressing experience of a missed diagnosis, hardly considered in the vast literature, is singled out in this inquiry. Poor judgment certainly does not explain the failures of serious physicians, therefore, the psychiatric viewpoint is applied, confining itself to experiences common to all physicians. The reported cases illustrated some danger points: Over-evaluation of spectacular symptoms, dramatic data of the history and the style and vogue of diagnostic and therapeutic fads of an impatient public for hasty decisions and the overselling popularizations of medical science. A clear differentiation of personal and impersonal factors in our work is indispensable. The craving for shortcuts, snap diagnoses, and conventional standardized fool-proof methods blind the insight into the unique

ness of each problem as new and individual. In a period where medical, legal, theological, and philosophical distress of the human sufferer merges into the request for help, the physician's challenge and position have widened immeasurably. To withstand the constant superhuman demands on his own emotional balance, he has constantly to reaffirm the two opposite forces within himself—resistance against outside pressures, and his enthusiastic impulse to help the sufferer and to learn the truth that "comes more readily from error than from confusion."

The Importance of Voice and Speech Pathology

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The development of oral communication enabled the evolution and propagation of human genius and culture. Keeping the socio-economic importance of voice and speech in mind, one can comprehend the detrimental influence of voice and speech disorders on all phases of human interrelationship. The study of voice and speech pathology is therefore interesting from many viewpoints.

Every function of oral expression is guided by central regulation. Intellectual performance represents the highest level. Memory patterns of sensory reception and motor expression are stored in the speech regions. The basal ganglia are concerned with emotional orientation and crude motor coordination of voice and speech, while the supranuclear neurons from cortex to bulbar nuclei provide association and conduction of afferent and efferent impulses. The cerebellum determines fine regulation of phonatory and articulatory movements. Bulbar affections demonstrate the influence of cranial nerve lesions on the motility of the speech organs.

Peripheral execution of speech depends on the interaction of pulmonary respiration, laryngeal phonation, pharyngeal sound modulation and oral articulation. Each phase has been studied by physiological methods. All vocal qualities like pitch, intensity or timbre represent physical phenomena. Among the theories of voice production, the "air puff" theory of passive vocal cord vibration in the expiratory air stream is the most probable. Phayrngeal sound modulation influences the esthetic qualities of the voice. Articulation produces vowels by selective oral resonance, and consonants by the addition of noises to the laryngeal voice at the articulatory narrowings of the various articulation zones.

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There are no true voice and speech organs, as voice and speech are secondary functions of organs for vital purposes: laryngeal occlusion during deglutition and expectoration, pharyngeal constriction and velar elevation during swallowing, as well as lingual, mandibular and labial movements for mastication. Palatal action determines the presence or absence of nasal resonance. Any interference with velar motility (cleft palate, congenital insufficiency, paralysis, injury etc.) produces nasal speech of rhinlalia aperta. The tongue's importance for speech is overrated. A rare "tongue-tie" (ankyloglossia) causes slight visible deviations of articulation without much audible distortion. Only the trilling "R" of foreign languages is impossible.

Pathological phonation may be studied by observing symptomatic dysphonias due to inflammations or tumors. Traumatic dysphonias due to laryngeal injury follow the same acoustical principles. Edema or infiltration of the vocal cords lowers vocal pitch. Their bowing favors falsetto voice. Loss of one vocal cord results in visibly irregular vibrations which explain the audible hoarseness. Voice therapy can relieve even complete aphonia by developing vicarious mechanism of phonation. Injuries to the external sternothyroid muscle may produce a post-operative dysphonia of the singing voice by disturbing the antagonistic equilibrium between this muscle and the cricothyroid. Esophageal speech serves to replace the laryngeal function following laryngectomy. Paralytic dysphonias are caused by paralysis of the superior or inferior laryngeal nerve. Exact examination reveals their different symptoms. Further differences are correlated with sudden onset or slow development of the lesions. Asymmetrical development of the larynx causes dysplastic dysphonias. Especially the congenital sulcus chordae vocalis, a horizontal furrow on the medial vocal cord edge, is important for the differentiation of chronic voice disorders.

The change of voice can be disturbed by several developmental dysphonias. In past times, the voice of the eunuch was purposely produced by castration before puberty. Eunuchoid voice represents a severe form of male hypogenitalism, while three forms of incomplete voice change are connected with slighter constitutional deviations. Precocious sexual development as a result of gonadic, adrenal and pineal disturbances or lesions below the third ventricle is accompanied by premature mutation. Perverse mutation of girls as seen in arrhenoblastoma of the ovary. Some perfectly healthy young men are unable to adjust to the low male voice and speak with habitual falsetto voice. This easily correctable disorder should not be confused with psychogenic falsetto voice due to a hysterical reaction, with the paralytic falsetto voice in laryngeal paralysis, or with the cerebral falsetto voice following cerebral concussion or encephalitis. Endocrine dysphonias of the adult comprise vasomotor monochorditis, menstural dysodia and laryngopathia gravidarum due to ovarian disfunction, and vocal disorders of thyroid, pituitary or adrenal origin.

Many voice disorders result from faulty speaking habits. Depending on the preponderance of excessive or

insufficient phonatory movements, these habitual dysphonias appear in hyperkinetic and hypokinetic types. An extreme form is the habitual false cord voice. Vocal misuse frequently leads to vocal cord nodes. Professional abuse of the voice causes phonasthenia, a progressive vocal fatigue complicated by secondary emotional reactions. A purely emotional origin is typical for the hyperkinetic and hypokinetic forms of psychogenic dysphonia, as seen in exhausted or emotionally maladjusted soldiers and hysterical women. Spastic dysphonia (psychophonasthesnia) represents a severe psychogenic disturbance characterized by interrupted and forceful phonation.

Infantile speech is frequently disturbed by developmental disorders. Delayed speech development results from birth injuries, prolonged disease or hereditary tendencies. Retardation of neuro-muscular maturation represents one cause of hearing mutism, while congenital auditory imperception seems to be connected with central lesions of acoustic perception. Similar handicaps are detected by the general and psychological examination of children who have difficulties with articulation (functional dyslalia). After these are overcome, agrammatism or reading disability may continue to interfere with progress in school. The characteristic otogenic dyslalias associated with deaf-mutism, hard of hearing speech and the speech of the deafened adult demonstrate the interrelationship between hearing and speech.

When the peripheral organs of articulation are impaired by malformations, injuries or paralysis, symptomatic dyslalias disturb normal pronunciation. According to the affected structures, labial, dental, lingual, palatal and nasal dyslalias produce typical deviations. Because of its practal importance, palatal dyslalia requires careful analysis. Velar motility may be disturbed by organic lesions or habitual inabilities which cause the corresponding organic and functional form of rhinolalia aperta. The opposite of this excessively nasal speech, namely lack of normal nasal resonance, may again be due to organic obstruction of the nose, or to a faulty speaking habit with excessive contraction of the velum. Thus, rhinolalia clausa also appears in an organic and functional variety. Open and closed nasality can coexist in four forms of mixed rhinolalia.

Incomplete speech development on the basis of mental retardation manifests itself in mental dyslogias. Typical abnormalities, as seen in cretinism, myxedema, mongoloidism or pituitary disease, complicate such speech disorders by additional endocrine dyslalias. While the motor (expressive), sensory (receptive) and amnestic (anomia) forms of dysphasia are generally known, the symptom of aphasic stuttering merits attention, because it shows that repetitions and hesitations may have an organic basis. Dysarthrias are explained by the size and localization of cerebral damage, and not by the causative disease itself. An anatomical description distinguishes pyramidal, extrapyramidal, frontopontine, cerebellar and bulbar dysarthrias.

Stuttering speech represents one symptom of dysphemia, a complex personality disorder with various neuro-vegetative and emotional manifestations. When the symptom

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of stuttering develops after cerebral concussions, it may be due to the corresponding endocranial lesions so that a syndrome of post-commotional dysarthria can be distinguished from genuine dysphemia. Another type of constitutional dysphrasia is called cluttering of which many patients are not aware, until they are urged to seek help. Psychogenic dysphrenias due to emotional shock or maladjustment exist in several forms: mutism with simultaneous loss of voice (aphonia), whispering (apsithyria) and articulation (anarthria); surdomutism occurs when functional deafness is superadded; hysterical phasia resembles motor dyphasia, but can be differentiated after careful observation; hysterical dysarthrias produce optically striking patterns of abnormal articulation; hysterical stuttering differs from the traumatic, aphasic and developmental forms of dysphemia with regard to onset, symptoms and prognosis.

CASE PRESENTATIONS

A 45 year old married woman came for a check-up to the Clinic first in May, 1945. As to the family history, her mother had died of cerebral hemorrhage in her early forties and two brothers who were twins had died immediately after birth. The applicant has no other brothers or sisters. She, herself, is childless; however, she had a still-born boy at the age of 29. Her only complaints are frequent headaches. She was 5 feet and 6 inches tall and weighed 135 pounds. The physical as well as the laboratory examination did not reveal abnormal findings.

Two years later this patient was admitted to the hospital. The history taken now was not in agreement with the history given on occasion of the first check-up. She stated this time that for several years before that first examination she felt insecure when walking; at times she staggered, particularly at night or when walking upwards or downwards; she noticed weakness in the extremities and she had difficulties in moving her fingers at will, particularly when writing; she also had observed that she had to void frequently and that occasionally she was even incontinent. Furthermore, she had been constipated for many years. She also admitted that her family physician had made a diagnosis of pernicious anemia and that she had received both liver injections and folic acid by mouth. The examination revealed a loss of weight of about 6 pounds in comparison with the first examination. The blood pressure was 120/70. The internal organs were negative. The blood count showed mild microcytic anemia (hemoglobin 64 per cent; R.B.C. 4,320,000; W.B.C. 6,800). The urine specimen showed a trace of albumin and 6 to 8 leuko-

cytes per hpf. The gastric analysis revealed 39° of total acid and 20° of free acid; after histamin test, the total acid rose to 69° after 15 minutes, to 75° after 30 minutes, to 86° after 45 minutes, and was 80° after 60 minutes. The spinal puncture showed a pressure of 125 cm water. The fluid was clear. The Queckenstedt test resulted in a raise of pressure to 152 bilaterally, dropping to 96. The Kahn text was negative, in both blood and CSF. The total protein was 11 mg. percent. The colloidal gold test of Lange gave a result of 1,110,000,000.

The neurological examination exhibited a beginning left optic atrophy. Of the deep reflexes, the biceps could not be elicited bilaterally; the knee tendon reflexes and the Achilles tendon reflexes were exaggerated on both sides. The Babinski was of the extensor type, bilaterally. There was a distinct ataxia in both arms and hands, and the gait showed lack of coordination. In both legs some spasticity could be noticed. The vibration sensibility was decreased in both lower extremities; the sense of motion and position was lost in two of the left toes.

The staff was of the opinion that the diagnosis made by the family physician was not correct. The blood count as well as the gastric juice analysis militated against pernicious anemia. Therefore, the neurological diagnosis of postero-lateral (combined) sclerosis could be abandoned. Most of the staff members had the impression that the closest diagnosis was multiple sclerosis. It was stresed that the neurological finding in the course of multiple sclerosis are changing and that the lack of the classical triad did not preclude the diagnosis. In this particular case there was no nystagmus and no intention tremor. The findings of the cerebro-spinal fluid also were not quite characteristic, and other symptoms, frequently present, such as diplopia and sensibility disturbances in the distal parts of the extremities were also not predominant in this case. One doctor stressed the fact that the abdominal reflexes were not absent in this case which would render the diagnosis of multiple sclerosis somewhat doubtful in his opinion. Another staff members pointed out that the history of this patient did not show the usual course of remissions and exacerbations, so typical for the majority of these cases; this patient's condition had apparently remained unchanged during the last decade. Another opinion was voiced that the age of onset was somewhat late in this case as generally the first symptoms of multiple sclerosis can be noticed before age 30.

While there was an agreement that the diagnosis was not substantiated without any doubt, the tentative diagnosis of multiple sclerosis was retained by the majority. (literature: G. A. Schumacher, J.A.M.A. 12:1059, July 22 and 13:1146, July 29, 1950—L. Hess, J. Nerv. and Ment. Dis. 1:30, January, 1950—T.J.C. von Storch and others, New York State Journal of Medicine 49:2145, 1945—P Phygesen, Arch Neurology and Psychiatry 4:339, April 1949—E. Bell, Jr., Journal of Insurance Medicine 1:20, December—January—February 1948—1949).

DIAGNOSTIC SUGGESTIONS

Basal Metabolism Rate (increased)

Conditions producing hypermetabolism: I. Endocrinopathy. a. Hyperthyroidism; b. Basophilic adenoma of the pituitary; c. adrenal cortex tumors; d. adrenal medullary tumors such as pheochromocytoma; e. diabetes insipidus; f. acromegaly. II. Blood Dyscrasia. a. leukemia-hypermetabolism usually occurs in the chronic type paralleling the number of white blood cells. Rates as high as 70% may be noted; b. polycythemia; c. anemia. III. Myocardial insufficiency—the metabolism may be elevated 50%. IV. anxiety state. V. Hypertension—increased metabolism may be as much as 30%. VI. Drugs. a. Thyrotoxicosis factitia; b. thyroxine; c. caffeine -7½ gr. of caffeine sodium benzoate may rise metabolism 25% in 2 hours. VII. Fever. VIII. Pregnancy. IX. Hemochromatosis. X. Technical Errors due to loss of oxygen. XI. Punctured Ear Drums (Wm. A. Stafne; L. E Wold and A C. Kohlmeyer. The Journal Lancet 10:378. October 1950)

Low Back Pain

The author quotes Caulk who stated that 80% of backaches in the male have their origin in the prostate and seminal vesicles, a fact frequently overlooked To distinguish between back pain of prostatic and non-prostatic origin four tests are recommended: 1)the prostatic massage test (relief after massage points to prostatic involvement); 2)the sound test (No. 20 or 22 French metal sound passed through urethra. If pain decreases after withdrawal of sound, prostate was responsible for back pain); 3)combined massage and sound test (interpretation as above) 4)novocain infiltration of prostate (panendoscope passed into urethra, and prostate is infiltrated with 1% novocain solution through a McCarthy needle. This test is the most reliable but causes more discomfort than the others). In the female low back pain may be caused by urethritis. Sound test and novocain infiltration of the bladder neck are differential diagnostic means. (E.F.Hock. J.Ins. Med. 4:42, Sept., Oct., Nov., 1950).

Essential Lymphocytophtisis

A new clinical entity has been reported the importance of which has yet to be evaluated. The authors stress that certain infectious diseases in adults and children lead, after absolute or relative lymphocytosis with strain of the lymphatic system beyond its capacity, to a more or less sudden decrease of the absolute number of lymphocytis, a sign which carries a serious prognosis. While this lymphopenia is generally due to an acute failure of the lymphatic system, lymphocytophtisis will develop more insidiously. It has been observed in tuberculosis of the lymph glands and spleen, in Hodgkin's disease, myelogenous leukemia and carcinomatosis of the lymph glands. One case of the latter category in a girl, age 15, with carcinoma of the epipharynx showed a terminal lymphocyte count of 49 per cu. mm. Two other cases of infants are presented, one with stubborn thrush associated with otitis media and bronchitis and an initial lymphocyte count of only 15 per cent which, before death fell to 2.5 per cent; the other one, also with thrush and preceding whooping cough (in which absolute lymphocytosis and strain on the lymphatic

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system is common) in whom progressive lymphocytophtisis developed. This child also died. The families of both children exhibited a pathologic gene of weakness of the hemotopoistic organs. (E. Glanzmann and P. Riniker. Annales paediatrici, 175:1, July-August 1950).

Diagnostic Suggestions

Diabetes and Emotional Stress

A rise in ketone bodies in the venous blood of both diabetics and non-diabetics takes place when stimuli out of the life experience of the patient and consciously or subconsciously felt as threats to his security become operative; the degree of ketone elevation is much higher in diabetics than in non-diabetics. These changes may lead to clinical acidosis, without alteration of insulin administration, intercurrent illness or change in physical activity. After disappearance of the stress situation and with the return of the feeling of security, the elevation of the blood ketone levels subsided without any medical intervention. (L.E.Hinkle, Jr.; G.B.Conger and St. Wolf J. Clin. Invest. 29:745, June 1950)

Diabetic Vulvitis

Pruritus vulvae is a common occurrence in diabetic women. It may persist with or without varying degrees of dermatitis secondary to scratching. Frequently a mycotic vulvitis or vulvovaginitis is superimposed and gives the characteristic picture of diabetic vulvitis: an inflamed, dark red or beefy appearance which first involves the vestibule and labia minora. and then spreads to adjacent parts. The high percentage of sugar in the secretions bathing the vulva is said to favor the growth of various fungi. As a result, irritations, excoriations and furuncles are common. (J. A. Gaines. Ciba Clinical Symposia. 6:179, July-August, 1950).

Acetylsalicylic Acid Poisoning

The symptoms vary from mild nausea, vertigo and tinnitus to collapse, unconsciousness, coma and death. Hospital records reveal the following symptoms: dizziness, tinnitus, deafness, vomiting, fever, prostration, rapid pulse, perspiration and in severe cases subnormal temperature, restlessness and coma. Aside of toxic reactions there also exists idiosyncrasy to the drug causing skin eruptions, edema, asthma, perspiration, palpitation, salivation, headache, vasomotor disturbances, syncope, collapse. A possible relationship between intensive salycilate medication and bleeding (prothrombinopenia) has been often mentioned in the literature. Of 88 thoroughly studied cases, slight intoxication was observed in 18; moderate in 22; severe in 17; fatal in 31 (most of which were suicides). (L. A. Greenberg. New Engl. J. Med. 243:124, July 17, 1950)

Lymphocytosis

Absolute lymphocytosis, in contrast to relative lymphocytosis often accompanying leukopenia, is found in the blood of infants and is also characteristic for certain infectious diseases, usually non-pyogenic, such as acute infectious lymphocytosis, infectious mononucleosis, and early stages of pertussis; it is also found in the secondary stages of mumps, German measles, undulant fever, tuberculosis and syphilis; further-

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more, in convalescence from acute pyogenic infections. Relative and absolute lymphocytosis accompany hyperthyroidism, especially when lymph node enlargement is present. (Wm. B. Castle. Disorders of the Blood. In: Wm. A. Sodeman, ed. Pathologic Physiology Mechanisms of Disease. Chapter 14; p.355. W. B. Saunders Co., Philadelphia, 1950)

Blood Pressure

As to blood pressure and clinical diagnosis, the following relations have been observed:

Bloodpressure	Diagnosis
Small pulse pressure with low systolic pressure	mitral stenosis, collapse, orthostatic collapse, acute heart failure.
High pulse pressure, with high systolic and conspicuously low diastolic pressure	aortic regurgitation, Graves disease, A-V anastomosis
Increased systolic and decreased diastolic pressure	aortic sclerosis, hypertension of old age, some (benign) forms of essential hypertension (peripheral arteries not yet narrowed)
Increased systolic pressure and more increased diastolic pressure	essential and renal hypertension.

(H. J. Sarre. Neue Med. Welt. 27/28:977, 1950)

SIDE GLANCES at the HISTORY OF MEDICINE

CURARE

Curare was first described by Peter Martyr in 1516. The first known medical paper on curare was prepared by B. C. Brodie. "Experiments and Observations on the Different Modes in Which Death is Produced by Certain Vegetable Poisons." Philos. Transact. 1811 p. 194 and 1812, p. 205. However, the scientist who discovered the pharmaceutical possibilities of mild curare poisoning was Claude Bernard. First he published a paper: "Analyse physiologique des propriétés des systèmes musculaire et nerveux au moyen du curare." Compt. rend. Acad. d. sc. 43:825, 1916, and then he treated the subject in his book: "Substances Toxiques," 1857, p. 344.

THERAPEUTIC SUGGESTIONS

Otitis Externa and Otitis Media

"The method used is to clean the ear thoroughly, using hydrogen peroxide to help loosen adherent particles. A snug cotton wick is inserted, and this is then saturated with 2% acetic acid and 10% Burrow's solution." Chronic external otitis usually cleared up in a few hours; for an acute inflammation it takes two to three days to improve. In a selected series of otitis media patients the author applied the following method: thorough cleaning of the ear with cotton applications, then hydrogen peroxide is instilled to further separate purulent debris from the middle ear cavity. The ear is then dried, and 5% acetic acid solution is instilled. Using a rubber ear syringe, alternate suction was applied in the manner of Proetz to permit the solution to enter any air spaces. With this technic the author has treated 38 ears and was successful in 32. (I. L. Ochs. J. Ins. Med. 4-44, Sept., Apr 29, 1950)

Migraine

The diagnosis must be secured and in individuals past 40 years of age aneurysm of the internal carotid artery must be ruled out. When a patient is seen during an attack of headache, a test may be performed in administering 0.25 mg. of ergotamine tartrate (gynergen) subcutaneously with repetition after half an hour. When the headache has been relieved ergotamine tartrate tablets (2 mg) to be taken every 15 minutes for a total of 5 doses, are precribed to be used in future attacks. In sudden painful seizures parenteral administration is more effective (Von Storch reported 90% favorable results). Ergonovine is less effective than gynergen, in some cases it may prove to be more successful when given orally. The maximum intravenous dose is 0.25 mg. and should bé limited to twice in 24 hours—not more than 6 every month. The maximum oral dose should not exceed 11 mg. in 24 hours —not more often than every second day and not more than 33 mg. a week. Peripheral vascular disease and coronary disease are contraindications. (J. J. Shea. The J. of the Omaha Mid-West Clin. Soc. 3:71, Aug. 1950)

Virus Hepatitis

Preventive measures: boiling in water (10-15 minutes) or autoclaving (5 minutes) of all needles and syringes prior to use; employment of both sterile needle and syringe for venipunctures; use of sterilized needles for blood counts; avoidance of multiple-dose-per-syringe inoculations. The treatment of the acute illness rests on bed rest and diet. The rationale of bed rest has received scientific support by S. E Bradley (Tr. Josiah Macy Jr. Foundation Conference on Liver Injury, 7:53, Jan. 1948; New Engl. J. Med. 240:456 March 24, 1949) who demonstrated that the hepatic blood flow diminishes when the patient assumes the erect posture or exercises. The use of high caloric diets with moderate fat restriction has been more empiric. Prohibition of alcoholic beverages is founded on the fact that they promote deposition of fat in the liver. The use of vitamins and lipotropic substances is not indisputably established. (S. Leibowitz. J. Ins. Med 4:10, Sept.-Nov. 1950)

THERAPEUTIC SUGGESTIONS

Tuberculosis of Traches

Paraamino salicylic acid (PAS) is effective against both streptomycin-senstive and streptomycin-resistant tubercle bacilli. Authors report on a case which illustrates the efficacy of PAS against tuberculosis of the trachea due to streptomycin-resistant tubercle bacilli. It is noteworthy that most of PAS was administered intravenously to avoid the gastroinestinal irritation, frequently complicating oral administration. (D. T. Carr; W. D. Schmidt: W. Herbert and A G Karlson. Minnesota Med 33:363, April 1950)—The effect of PAS may be enhanced by premedication of p-benzoic acid (commercial name: Benemid); 0.5 gm of Benemid every 6 hours for 24 hours followed by 4 gm. of PAS. Administration of this combination for three weeks daily not accompanied by toxicity. (W. P. Boger and F. W. Pitts. Science 112:149, 1950)

Khellin (Visammin)

A coronary vasodilatator obtained from Ammi visnaga, a plant of the Umbelliferae family. Of 250 cases of angina pectoris, 56% responded well, 34% moderately, 10% did not respond. In 50 cases of myocardial infarction no conclusions were reached. In treatment of asthma, results were good although dosage had to be higher than in angina pectoris. (L. D. Diaz. Bol. del Inst. de Patologia med. 5:63. April 1950)—H. A. Dewar and T. A. Grimson, in the Brit. Heart J. 12:54, 1950, report favorable results in preventing angina in 12 cases.—R. H. Roseman et al. (J.A.M.A. 2:160, May 13, 1950) came to similar conclusions.

Eye Infections

Sodium propionate has been used by the author in about 1,200 cases of conjunctivitis, blepharitis and keratitis of various forms and degrees. A recent evaluation of 238 cases in which cultures, smears and epithelial scrapings were done showed no improvement in only 5 instances of 130 cases of acute bacterial conjunctivitis, in only 1 case of 18 cases of chronic conjunctivitis, in 2 of 50 cases of chronic blepharoconjunctivitis, in 1 of 10 cases of other chronic lid infections and in 2 of 2 cases with corneal ulcer. A 5% solution of sodium propionate has been applied; side effects or sensitivity to the drug are rare. (F. H. Theodore. J.A.M.A. 3:336, May 20, 1950)

Medication Before Anesthesia in Children

To avoid mucus secretion during tonsillectomy and adenoidectomy scopolamine has been used in preference to atropine. The identical dosage of scopolamine and atropine was administered of each drug, and that 0.0002 gm. from age two to five 0.0003 gm from age six to ten and 0.0004 gm from age eleven to fourteen. The injection was made half an hour to one hour before anesthesia with Vinethene ether was started. Mucus was absent in 73.2 percent of the scopoloamine treated cases and in 49.3 percent of the atropine treated cases. It also appears that the sedative effect of scopolamine is helpful in inducing anesthesia. (J. S. West and E. M. Papper. Anesthesiology, 11:279, 1950)

BOOK REVIEWS

Explorer of the Human Brain

The Life of Santiago Ramon Y Cajal. By Dorothy F. Cannon. Henry Schuman, New York, 1950. 363 pages. Cloth. Price, $4.00.

The life history of Ramón y Cajal is stimulating not only because he was an outstanding investigator of the histology of the central nervous system but, much more, because histology was his way to approach the complex mysteries of life. The dynamic polarization of the neuron, the one-directional progress of the nervous impulse, the individualistic and, simultaneously, associative behavior of the neuron (all concepts which he derived, and proved by new staining methods, from interpretations of microscopical pictures) afforded him the physiological means of drawing closer to the enigmas of existence which he obviously intended to tackle in his last years of life, according to the titles of some lost manuscripts, such as "Notes on Histological Psychology" and "Mystery of the Tomb and Hallucinations and Dreams".

From a characterological angle, the eminence of Ramón y Cajal's personality must be comprehended by the wise and controlled restriction of his work to a small sector of biology which he, finally, could build up and enlarge to become a vantage point from which to view the totality of somato-psychic phenomena. This judicious restraint earmarks the genius, as mastery, according to the German poet Schiller, unfolds itself in critical self-limitation.

Miss Cannon's presentation of both life and work of Ramón y Cajal is impressive and adequate. At times, particularly in the first part of the book, this reviewer had the feeling that too much weight was given to non-essential matters and details which would rather dim than clarify the biography of this distinguished scientist's development; this reviewer, likewise, could not quite agree with the wording of the headings of the chapters which do not seem to harmonize with the stern objectives of Cajal's struggle, aspirations, and achievements.

Yet, in the whole, this book gives a vivid, lucid, and clearly drawn description of the human and scientific uniqueness of the greatest medical scholar that Spain had ever brought forth. F.R.S.

Textbook of Endrocrinology

Edited by Robert H. Williams. W. B. Saunders Company. Philadelphia-London, 1950. 793 pages. Cloth.

This new textbook has been prepared by twelve authors. The first chapter, written by the editor, sets forth in a very instructive manner the general principles of endocrinology and throws clearly into relief the progress in this branch of medicine as achieved in the past decades. The following chapters deal with the endocrine glands (pituitary, thyroid, adrenals, testes, ovaries, pancreas-diabetes mellitus, parathyroids). This reviewer was impressed by the excellent presentation of the difficult problem: neuro-endocrine and psychodynamic as pects of the endocrinopathies, by Harry B. Friedgood. The chapter on obesity by L. H. Newburgh will be of special interest for insurance physicians. The illustrations, particularly those concerned with diagnostic features, are well selected and contribute to the lucidness of the description. F.R.S.

BOOK REVIEWS

Victory Over Pain

A History of Anesthesia, by Victor Robinson M.D., Henry Schuman, New York, 1950. Cloth. 338 pages. 48 illustrations. $3.50.

Dr. Robinson, Professor of History of Medicine at Temple University and founder of the first monthly journal of medical history in English language, 'Medical Life', has, indeed, the experience and scientific background to write a book on the history of anesthesia and analgesia. The presentation starts with prehistoric times and leads through the milleniums to the most recent developments in the fight against pain. It is interesting to learn, that already Dioscorides in the first century A.D. knew about opium as "a pain easer and a sleep-causer, and that refrigeration anesthesia (by the use of snow) was introduced already in the middle of the 17th century by Marco Aurelio Severino of Naples. With the 7th chapter of his book, the author turns from the description of events to the historical biography of champions of anesthesia: Humphrey Davy (nitrous oxide), Henry Hill Hickman (Carbon Dioxide), Franz Anton Mesmer (hypnotism), Crawford Williamson Long (ether), Charles Thomas Jackson (ether), William Thomas Green Morton (ether in dentistry), Samuel Guthrie in U. S., Soubeiran in France, and Liebig in Germany (chloroform), Benjamin Ward Richardson (ether spray, the beginning of local anesthesia), Carl Koller (cocaine, local anesthesia in eye surgery), William Stewart Halstedt (cocaine, conduction anesthesia), James Leonard Corning (spinal anesthesia), Heinrich Braun (novocain), Carl Gauss (twilight sleep), A. Hingson and W. B. Edwards (continuous caudal analgesia), Karel Maydl, Victor Eisenmenger and Franz Kühn (endotracheal anesthesia), Pirogoff (1847), Daniel Mollière (1884), J. H. Cunningham and F. H. Lahey (1903) (rectal anesthesia), Robert Boyle (1656), Sigmund Elsholtz (1665), Pierre-Cyprien Oré (1872), Wise and Sharpff (1932) (intravenous anesthesia). Cyclopropane was discovered by Lucas and Henderson in 1929. Ethylene was introduced by Luckhardt and Carter in 1923. Curare was noticed first by Antonio Pigafetta in the 16th century. Curarine, the alkaloid of curare, was discovered by Boussingault and Roulen in 1828; finally H. R. Griffith and G. E. Johnson introduced curare into anesthesia in 1942. This short resume may give a superficial impression of the scope of this work. It is a moving document of man's incessant struggle against pain which, paradoxically enough is given by nature "to Life for its protection." And yet, as Robinson has begun his "The Story of Medicine" (1931): "The first cry of pain through the primitive jungle was the first call for a physician."

F.R.S.

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